

# Non-surgical septal reduction by coil embolization in patients with hypertrophic obstructive cardiomyopathy. An alternative to alcohol ablation

Przezskórna redukcja gradientu w drodze odpływu lewej komory metodą embolizacji tętnicy przegrodowej mikrokoilami u osób z kardiomiopatią przerostową zawężającą

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## Abstract

Dynamic obstruction of the left ventricular outflow tract is present in 25-30% of patients with Hypertrophic Cardiomyopathy (HCM). The non-surgical method of interventricular septum thickness reduction using coils is a new technique of percutaneous treatment in patients with hypertrophic obstructive cardiomyopathy (HOCM). The presented method could be an interesting alternative for left ventricular outflow tract pressure gradient reduction in patients with HOCM in whom alcohol ablation may be related to a higher risk of complications. We present a description of two patients with HOCM and drug-refractory symptoms, who underwent closure of the septal branch using coils in the Institute of Cardiology.

**Key words:** hypertrophic obstructive cardiomyopathy, coil, embolisation, alcohol ablation

## Streszczenie

W grupie pacjentów z kardiomiopatią przerostową (ang. *hypertrophic cardiomyopathy*, HCM) u 25–30% występuje dynamiczne zawężanie w drodze odpływu lewej komory (ang. *left ventricular outflow tract*, LVOT). Nową, opisaną zaledwie kilka lat temu, propozycją przezskórnego leczenia osób z kardiomiopatią przerostową zawężającą (ang. *hypertrophic obstructive cardiomyopathy*, HOCM) jest metodą nieoperacyjnej redukcji grubości przegrody międzykomorowej za pomocą koili naczyniowych. Prezentowana metoda może stanowić interesującą alternatywę redukcji gradientu w LVOT u pacjentów z HOCM, u których ablacja alkoholowa jest obarczona zbyt dużym ryzykiem wystąpienia powikłań. Przedstawiamy opis 2 osób z HOCM, u których wykonano zabieg zamknięcia tętnicy przegrodowej za pomocą koili w Instytucie Kardiologii w Warszawie.

**Słowa kluczowe:** kardiomiopatia przerostowa zawężająca, koil, embolizacja, ablacja alkoholowa

## Introduction

Dynamic obstruction of the left ventricular outflow tract (LVOT) is present in 25-30% of patients with hypertrophic cardiomyopathy (HCM) [1]. Pressure gradient in LVOT > 50 mmHg caused by the obstruction is related to faster progression of heart failure symptoms, to increased risk of

sudden cardiac death and to increased frequency of implantable cardioverter-defibrillator (ICD) interventions [2-4]. There are usually three therapeutic options in patients with hypertrophic obstructive cardiomyopathy (HOCM): pharmacotherapy as the first choice and as a second choice surgical myectomy or percutaneous septal alco-

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hol ablation [5]. Qualification for invasive treatment is limited to patients with no improvement of symptoms on pharmacotherapy (remaining in class III or IV according to NYHA) and with a resting LVOT gradient  $\geq 50$  mmHg [6, 7].

Alcohol septal ablation (ASA) is a non-surgical method of basal interventricular septum thickness reduction. This procedure was done for the first time in 1994 in London by U. Sigwart. Alcohol septal ablation remains the most frequently performed intervention in patients with HOCM [8]. The procedure leads to gradient reduction in LVOT as a consequence of post-myocardial infarction remodelling of the left ventricular muscle [9-13].

The non-surgical method of interventricular septum thickness reduction using coils is a new technique of percutaneous treatment in patients with HOCM, described for the first time a few years ago. There is a small number of published reports on the use of coils for that indication despite their routine use for closure of intracranial aneurysms and congenital vascular malformations. There is an ongoing discussion on the use of coils as an alternative to alcohol ablation in a selected group of patients with HOCM [14, 15].

We present a description of two patients with HOCM who underwent closure of the septal branch using coils in the Institute of Cardiology.

## Case reports

### Case 1

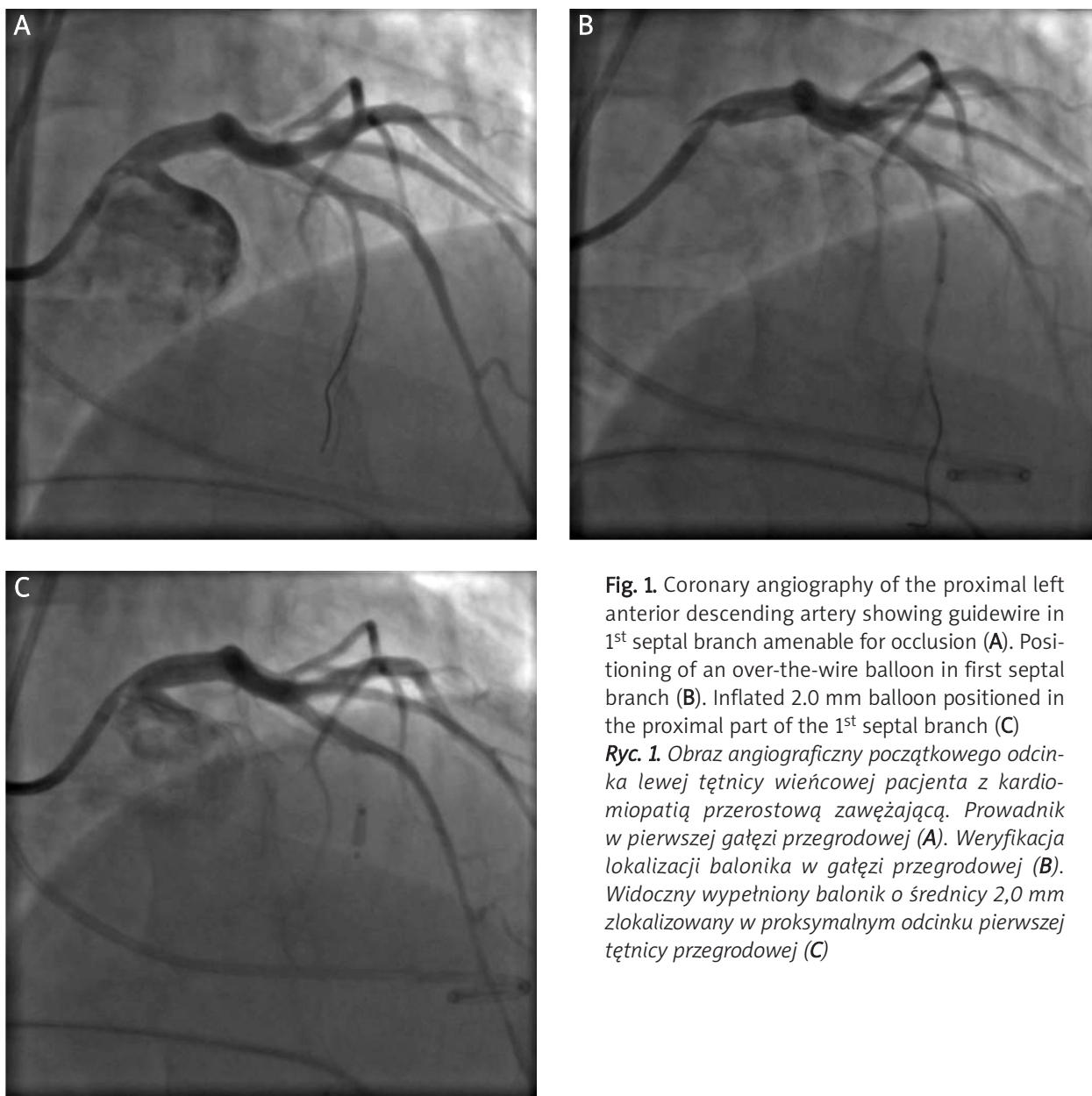
A 47-year-old man with hypertrophic cardiomyopathy diagnosed in 2006 and treated pharmacologically since then with  $\beta$ -blockers (metoprolol 200 mg/day) and calcium channel blockers (verapamil 20 mg/day) was admitted to the Institute of Cardiology for elective invasive treatment – closure of the 1<sup>st</sup> septal branch originating from the left anterior descending artery (LAD) of the left coronary artery (LCA). In 2008 he underwent ICD implantation because of ventricular arrhythmia. On admission he was in NYHA functional class III. Electrocardiogram performed in the Institute of Cardiology during hospitalization showed regular sinus rhythm with signs of left ventricular hypertrophy. Echocardiographic examination demonstrated systolic movement of the anterior mitral valve leaflet (systolic anterior movement – SAM), maximal resting LVOT gradient of 140 mmHg, basal interventricular segment thickness of 26 mm and posterior left ventricular wall thickness of 23 mm. Direct pressure measurement performed on heart catheterization disclosed a maximal resting gradient of 80 mmHg. There were no significant lesions in coronary arteries on coronary angiography. The patient was disqualified from septal alcohol ablation during previous hospitalization due to the presence of multiple fistulas draining to the right ventricle after contrast injection into the 1<sup>st</sup> septal branch seen on both coronary angiography and transthoracic echocardiography. Therefore there was a justified fear that the absolute alcohol injected into the artery

would not cause its chemical obliteration but would leak to the right ventricular cavity and to the pulmonary circulation. After talking to the patient informed consent was obtained for septal branch embolization using coils. The procedure is performed percutaneously using an over-the-wire balloon catheter which is introduced into the proximal part of the chosen septal branch (figs. 1A-1C). The balloon is usually 10 mm long and its diameter is adjusted to the size of the artery. To better delineate the area of interventricular septum supplied by the chosen artery the echocardiographic contrast Sono Vue is injected through the internal lumen of the catheter before coil implantation. The contrast-enhanced basal part of the interventricular muscle is then identified using intraprocedural transthoracic echocardiography. As in the previous study there was a contrast leak to the right ventricular cavity and pulmonary artery. After identification of the correct septal branch a microcatheter (Cook Inc.) was introduced into its lumen and used to deliver two platinum coils of 60 mm and 40 mm length and 3 mm diameter (Cook Inc.), obliterating the artery lumen (figs. 2A, 2B).

There was a TIMI 1 flow in the 1<sup>st</sup> septal branch directly after the procedure. An immediate LVOT gradient reduction from 80 mmHg to 30 mmHg was confirmed in direct assessment. Due to chest pain which appeared after artery closure 5 mg of morphine was administered. Femoral artery access was closed with the StarClose system (Abbott Vascular). The patient was monitored in the Intensive Care Unit (ICU) for the following 48 h. Troponin I concentration 12 and 24 h after the procedure rose to 3.95 ng/ml and 3.23 ng/ml respectively and CK-MB to 14.7 U/l and 7.0 U/l respectively. Echocardiography performed 5 days after the procedure showed a maximal resting LVOT gradient of 112 mmHg. The patient was discharged home in a good general condition 8 days after the procedure. Transthoracic echocardiography performed 6 weeks after the procedure showed a maximal resting LVOT gradient of 110 mmHg. Computed tomography angiography of the coronary arteries confirmed occlusion of the 1<sup>st</sup> septal branch. There was improvement of symptoms as assessed on follow-up visits 3 months and 6 months after the procedure and there was a persisting echocardiographically measured maximal resting LVOT gradient of 115 mmHg. Therefore the patient was qualified for surgical myectomy.

### Case 2

A 53-year-old man with obstructive hypertrophic cardiomyopathy diagnosed in 2005 and treated pharmacologically since then with  $\beta$ -blocker (bisoprolol 10 mg/day) was admitted for elective invasive treatment. The patient was qualified for embolization of the 1<sup>st</sup> septal branch during previous hospitalization. Electrocardiogram performed on admission showed regular sinus rhythm with signs of left ventricular hypertrophy and ST-segment depression in precordial leads characteristic for hypertrophic cardiomy-



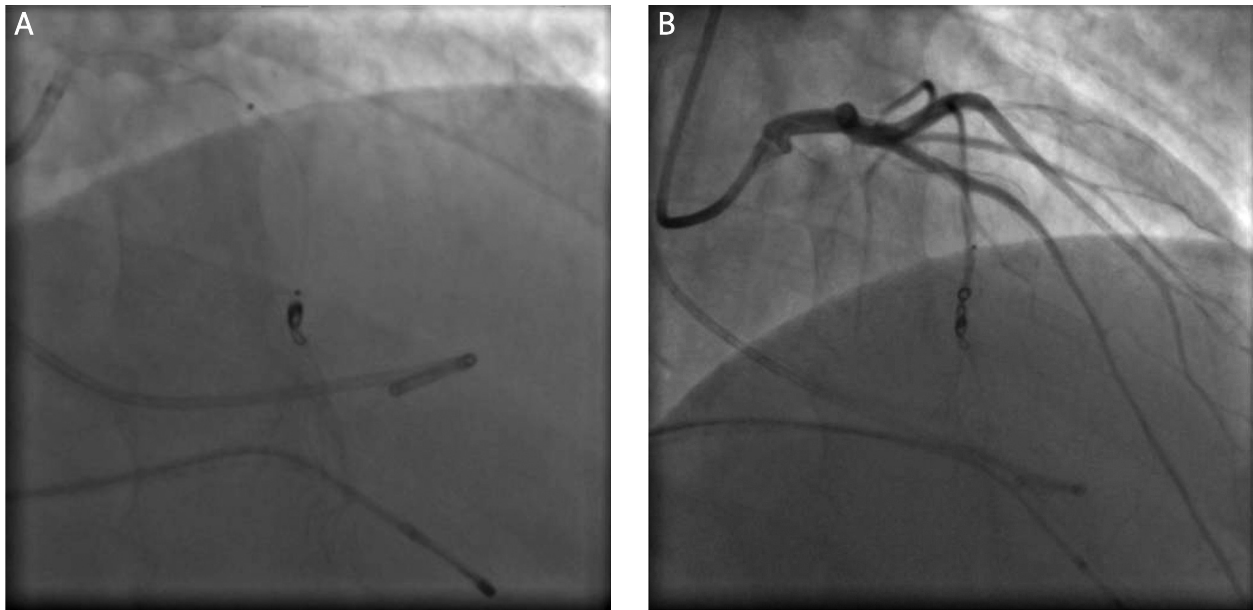
**Fig. 1.** Coronary angiography of the proximal left anterior descending artery showing guidewire in 1<sup>st</sup> septal branch amenable for occlusion (A). Positioning of an over-the-wire balloon in first septal branch (B). Inflated 2.0 mm balloon positioned in the proximal part of the 1<sup>st</sup> septal branch (C)

**Ryc. 1.** Obraz angiograficzny początkowego odcinka lewej tętnicy wieńcowej pacjenta z kardiomiopią przerostową zawężającą. Prowadnik w pierwszej gałęzi przegrodowej (A). Weryfikacja lokalizacji balonika w gałęzi przegrodowej (B). Widoczny wypchnięty balonik o średnicy 2,0 mm zlokalizowany w proksymalnym odcinku pierwszej tętnicy przegrodowej (C)

opathy. Echocardiographic examination done at the time of qualification for the procedure demonstrated SAM, maximal resting LVOT gradient of 98 mmHg, basal inter-ventricular septum segment thickness of 17 mm and posterior left ventricular wall thickness of 16 mm. Direct pressure gradient measurement on cardiac catheterization revealed maximal resting LVOT gradient of 70 mmHg. There were no significant lesions in coronary arteries on coronary angiography. Coronary angiography performed during qualification for alcohol septal ablation with simultaneous echocardiographic examination demonstrated the leak of contrast medium into the left ventricular cavity after selective contrast injection into the 1<sup>st</sup> septal branch. The inflow of contrast medium into the left ventricular cavity suggested the presence of communication between the

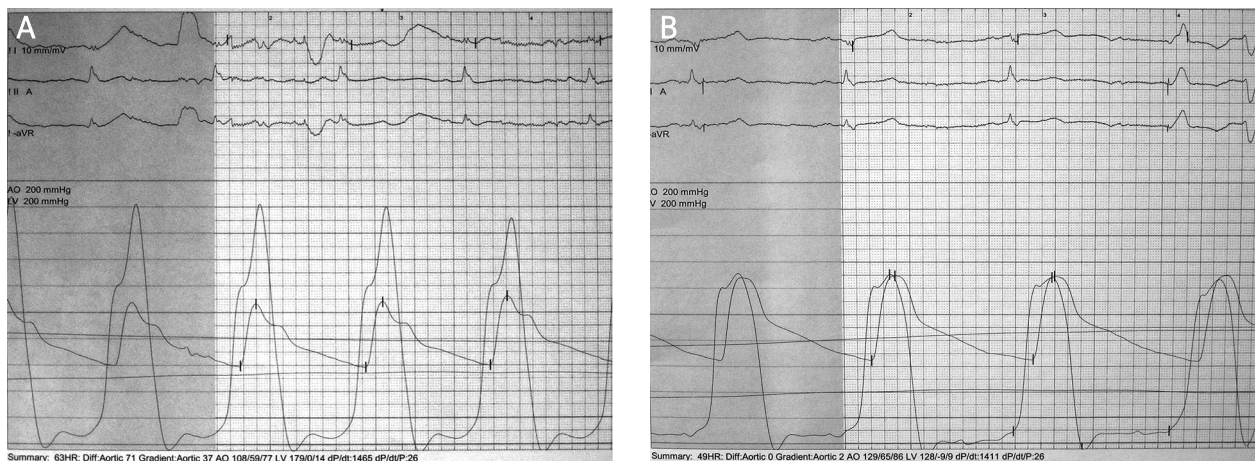
septal branch and the left ventricular cavity and a justified risk of absolute alcohol leak to the left ventricle. Therefore the patient was not qualified for alcohol septal ablation. Because of a persistent worsening of heart failure symptoms (NYHA class III) despite modification of the pharmacotherapy, the presence of an LVOT pressure gradient and the lack of patient's consent for surgical treatment, obliteration of the 1<sup>st</sup> septal branch using coils was suggested. The patient gave written consent for the treatment.

The procedure of 1<sup>st</sup> septal branch closure was performed via right transfemoral access using a microcatheter (Cook Inc.) and two platinum coils of 40 mm length and 3 mm diameter (Cook Inc.). TIMI 0 flow in the artery was achieved and the LVOT pressure gradient dropped from 70 mmHg to 5 mmHg on direct assessment (figs. 3A, 3B). Throughout



**Fig. 2.** Coronary angiographic projection of the left anterior descending artery with first septal branch during (A) and after (B) coil embolization

**Ryc. 2.** Obraz angiograficzny lewej tętnicy wieńcowej w trakcie (A) i po (B) embolizacji pierwszej gałęzi przegrodowej koilami



**Fig. 3.** Presence of the left ventricular outflow tract gradient at rest before (A) and substantial reduction after (B) alcohol septal ablation procedure

**Ryc. 3.** Zapis jednoczesnego pomiaru ciśnienia w lewej komorze i aorcie wstępującej. Bezpośrednio przed zabiegiem ASA gradient spoczynkowy wynosi ok. 70 mmHg (A). Po skutecznym zabiegu ASA uzyskano redukcję gradientu do ok. 5 mmHg (B)

the whole procedure the patient did not complain of chest pain. Femoral access was closed with the StarClose occluder (Abbot Vascular). The patient was monitored in the ICU for the next 48 h. There was an increase of troponin I concentration to 3.16 ng/ml and 3.49 ng/ml and CK-MB to 10.2 U/l and 8.2 U/l at 12 h and 24 h after the procedure, respectively. Follow-up echocardiography done 6 days after the procedure showed a persistent resting LVOT gradient of 92 mmHg. The patient was discharged home in a good general condition 6 days after the procedure.

During follow-up visits after 3 and 6 months there was a progressive improvement of exercise tolerance (to NYHA class I/II and I, respectively). Echocardiographic examinations performed after 3 and 6 months showed a reduction of the maximal LVOT gradient to 27 mmHg and 19 mmHg, respectively.

## Discussion

Successful alcohol septal ablation in most cases leads to reduction of the basal interventricular septum thickness

and to permanent reduction of the LVOT gradient. Significant improvement of symptoms is observed in 90% of patients who undergo the ASA procedure. In patients who are correctly qualified for the procedure ASA leads to necrosis and subsequently fibrosis of the basal segment of the interventricular septum. Selective contrast medium administration into the septal branch under echocardiographic control preceding the procedure allows a detailed assessment of the localization and borders of the myocardium supplied by the artery. This verification is necessary for the final qualification of the patient for ASA. The authors of the few published reports on the use of coils for LVOT pressure gradient reduction suggest that this technique leads to the creation of a "better controlled" myocardial infarction in comparison to ASA [14, 16]. The term "better controlled" is related to limitation of the necrosis to the area of ischaemia defined by the contrast echocardiography performed before the procedure. Alcohol injection during ASA leads to a different mechanism of myocardial infarction where ischaemia is accompanied by denaturation of the cardiomyocyte proteins caused by the diffusion of alcohol through the vessel wall, causing less predictable and more severe necrosis. However, it seems that the extent of necrosis assessed on magnetic resonance imaging after obliteration of the artery with coils is smaller than expected, which may influence the effectiveness of those procedures [11-13, 17-27].

The interventricular septum is a well arterialized muscular structure with a potent network of collaterals. Closure of the septal branch with coils leads to ischaemia, but does not always cause necrosis of the whole area of the interventricular septum segment defined by the contrast echocardiography. The extent of necrosis depends not only on the diameter of the obliterated septal artery, but also on the degree of collateralization of the interventricular septum muscle provided by the right coronary artery and other septal branches. Therefore an initially significant LVOT gradient reduction observed in patients with good septal arterialization may progressively vanish on long-term follow-up with ongoing improvement of the contractility of stunned septal myocardium weeks or months after the implantation of coils. This may potentially explain why in one of the presented cases despite LVOT gradient reduction directly after the procedure and the confirmation of artery occlusion on computed tomography there was no clinical improvement with ongoing increase of the gradient to significant values on the follow-up echocardiographic examinations. The problem of a lower efficacy of coils in comparison to ASA was discussed by Durand *et al.*, who reported that among 20 patients undergoing septal branch obliteration with coils permanent LVOT gradient reduction below 50 mmHg was not achieved in 25% of patients after 6 months of observation [14]. Necrosis caused by ASA is determined by two factors: ischaemia and cytotoxic influence of the alcohol on cardiomyocytes.

Therefore the degree of arterialization and collateralization of the septum have a significantly lower influence on the area of permanent myocardial injury in comparison to the implantation of coils [27]. It seems that this is the reason for the significantly greater extent of necrosis and a higher creatinine kinase concentration after ASA in comparison to septal branch embolization with coils as observed by Durand *et al.* [14].

Higher efficacy of the ASA procedure defined by permanent LVOT gradient reduction may be related to the higher risk of complications. The most frequent ones include ventricular arrhythmias and atrioventricular conduction abnormalities up to a complete block. Less frequent complications include iatrogenic interventricular septum rupture, papillary muscle rupture and acute mitral regurgitation, acute anterior myocardial infarction caused by alcohol leak from the septal branch to the LAD, coronary artery dissection and pericardial tamponade [3, 8].

The risk of transient or permanent conduction abnormalities after alcohol septal ablation is related to the volume and localization of the myocardium supplied by the occluded artery as well as by the amount of alcohol injected into the septal branch [28-32]. There is a positive correlation between extent of the necrosis caused by ASA and the frequency of conduction abnormalities during and after the procedure [33]. The occurrence of permanent conduction block is probably related to the diffusion of alcohol through the capillary system and requires pacemaker implantation in 10-15% of cases [14, 17].

Despite the use of lower amounts of alcohol (1.5-2 ml) and its slower injection into the artery with prolonged 10 min inflation of the balloon occluding the septal branch, which reduced the ASA complications, especially those related to conduction abnormalities, there is still a group of patients qualified for ASA in whom the risk of described complications is higher and may be a contraindication for this procedure [28, 30-32]. This group includes patients with atrioventricular conduction abnormalities present before the procedure or with a large septal branch penetrating to the lower parts of the interventricular septum or covering the whole thickness of the myocardium [34].

Embolization of the septal branch with a coil leads to ischaemia and subsequent necrosis of the chosen muscle fragment without a direct, toxic influence of the alcohol on the conduction system. It is probably related to a lower efficacy of the procedure, but also to a lower risk of complications such as conduction abnormalities and especially to a lower frequency of permanent atrioventricular blocks [18, 35-37].

Therefore we think that patients at a higher risk of atrioventricular block after alcohol injection into the septal branch should be considered for percutaneous septal branch obliteration with coils if indicated. Another group of patients with hypertrophic obstructive cardiomyopathy requiring invasive treatment but not qualified for ASA

includes those with angiographically or echocardiographically confirmed communication between the septal branch chosen for obliteration and other vessels or heart cavities [14, 18, 27].

Both presented cases represent the latter group of patients. In these situations ASA can lead to the alcohol leaking out of the septal branch into the pulmonary or systemic circulation with not clearly known, but potentially severe complications. According to our knowledge the described cases represent the first use of coils for the treatment of HOCM in Poland. In our opinion the described method is an interesting alternative for the treatment of LVOT pressure gradient in patients with hypertrophic obstructive cardiomyopathy in whom alcohol ablation may be related to a high risk of complications. A few studies presented so far indicate that this method may be less effective, but it is also related to a lower risk of permanent conduction abnormalities in comparison to alcohol ablation.

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